TABLE 1. Demographic and Clinical Characteristics of 68 Hospitalized Children With RSV Bronchiolitis Divided Into Wheezing-positive and Wheezing-negative Groups

Item	Wheezing Positive (29)	Wheezing Negative(39)	*P*
Sex (M/F)	17/12	26/13	NS
Age (d)	59.5 ± 34.1	66.9 ± 55.5	NS
Eosinophils (cells/μL)	132.1 ± 164.4	80.0 ± 87.4	0.045
Viral load	$4.0 \times 10^8 \pm 1.1 \times 10^9$	$1.1 \times 10^7 \pm 2.3 \times 10^7$	0.015
IFN-λ1	5.3 ± 5.5	2.6 ± 4.3	0.065
IFN-λ2/3	2.9 ± 4.6	1.1 ± 1.4	0.047

M/F indicates male/female; SD, standard deviation; NS, nonsignificant.
Values are expressed as mean ± SD.

The most significant result of our study is that we found substantial evidence of the correlation between a high RSV-RNA load and recurrent wheezing in former bronchiolitis children. This finding seems to be unrelated to the clinical severity of the bronchiolitis. A possible explanation of the mechanism through which RSV infection could increase susceptibility to wheezing in children is the chronic epithelial reactivity changes in the still immature and developing lung of young infants.[1] Our study results support the hypothesis that viral factors may play a role in the airway sequelae possibly by directly amplifying the immune response of the host. Nevertheless, the hypothesis that children prone to wheezing are also prone to have high viral loads cannot be ruled out. The long-term consequences on lung function might be due to the airway remodeling resulting from virus-induced inflammation, along with efforts to repair airway damage. Unfortunately, even if it usually resolves the infection, sometimes it causes enhanced pathology.[4]

Type I IFNs are cytokines crucial for antiviral resistance. In 2003, a novel class of antiviral cytokines was discovered, characterized and classified as type III IFNs (λ), whose antiviral properties were similar to those of type I IFNs but appeared to be expressed especially by epithelial cells.[10] As expected, we found a coordinate expression of the different subtypes of IFN-λ. Moreover, a correlation between RSV-RNA load and IFN-λ expression was demonstrated. A high IFN-λ level was found in children with recurrent wheezing after RSV bronchiolitis. We could speculate that in wheezing children, the activation of IFN-λ2/3 as well as of IFN-λ1 during bronchiolitis was ineffective in controlling the high viral load. Unfortunately, on the basis of our study results, we could not conclude whether the viral load was high because IFN-λ was activated but "deficient" or whether because of the high viral load, IFN-λ was not able to contain the infection. The demonstration that a high dose of IFN-λ1 is required for effect[11] seems to make the hypothesis of an IFN-λ activated but "deficient" the most likely. Moreover, studies on RSV-infected airway epithelial cells from children with wheezing demonstrated an intrinsic IFN-independent defect in the antiviral response that resulted in elevated viral infection.[12]

A distinct point of our study is the adherence to strict inclusion criteria in bronchiolitis diagnosis, such as age <1 year, infants with the first episode of lower respiratory infection and the presence of crackles, exclusion of infants with wheezing in whom early presentation of asthma can overlap the bronchiolitis.

A possible limitation of our study could be the nature of the interview performed via telephone.

In conclusion, we demonstrated that a high RSV-RNA load and IFN-λ activation at the time of admission for bronchiolitis are associated with recurrent wheezing at 36-month follow-up.

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MENINGOMYELORADICULITIS AS AN UNUSUAL PRESENTATION OF NEUROBORRELIOSIS IN CHILDHOOD

Maria Vânia Sousa, MD, Isabel Lopes de Carvalho, PhD,† Maria Sofia Núncio, MD,† Carla Conceição, MD,‡ Rita Silva, MD,§ and Catarina Gouveia, MD**

Abstract: We report a pediatric case of Lyme neuroborreliosis–associated meningomyeloradiculitis with atypical manifestations and negative initial cerebrospinal fluid borrelial antibodies. Transverse myelitis and painful radiculoneuritis have rarely been described in pediatric neuroborreliosis. Clinical manifestations are wide ranging and nonspecific, and the serologic diagnosis is often delayed in the acute phase.

Key Words: *Borrelia burgdorferi* s.l, neuroborreliosis, transverse myelitis, meningomyeloradiculitis

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From the *Division of infectiology, Department of Pediatrics, Hospital Dona Estefânia; †National Health Institute Doutor Ricardo Jorge; ‡Department of Radiology, and §Division of Child Neurology, Department of Pediatrics, Hospital Dona Estefânia, Lisboa, Portugal.
The authors have no funding or conflicts of interest to disclose.
Address for correspondence: Maria Vânia Sousa, Hospital Dona Estefânia, Rua Jacinta Marto, 1169-045 Lisboa, Portugal. E-mail: mvtsousa@gmail.com

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Lyme borreliosis is a multisystem disease with a wide range of clinical manifestations. Neurologic involvement occurs in 5–10% of patients, affecting either the peripheral or the central nervous system (CNS).[1] The most common presentation in children is acute facial

nerve palsy (55% of patients). In general, the clinical characteristics of childhood and adult neuroborreliosis are similar, except for meningopolyradiculoneuritis, which is unusual in children. Lyme transverse myelitis is also a severe, uncommon presentation in children.[2,3]

We report a child with Lyme meningomyeloradiculitis who presented atypical manifestations and remarkable clinical and imaging dissociation.

CASE PRESENTATION

A previously healthy 11-year-old boy was admitted for severe frontal headache and a diffuse intense nonradicular cervical pain for 3 months. Additional complaints included intermittent low-grade temperature, fatigue and vomits. No history of rash, tick bites, recent immunizations or exposures to infectious diseases was reported. Three months before, the patient had traveled to Moldova and a rural area of south Portugal.

On examination, he was apyrexial with nuchal rigidity and cervical hyperesthesia but his general and neurological examination was otherwise unremarkable.

A complete blood cell count and inflammatory markers were normal.

Spinal cord magnetic resonance imaging (MRI) revealed an abnormal T2 hyperintensity within the spinal cord from D2/D12, with gadolinium enhancement at the periphery in several cervical and dorsal roots. These changes were suggestive of a widespread transverse myelitis with polyradiculitis. Brain MRI was normal and there was no cerebral or meningeal enhancement.

The cerebrospinal fluid (CSF) revealed mild pleocytosis (200 cells/mm^3, lymphocytes), elevated protein (182.2 mg/dL) and hypoglycorrhachia (ratio: 0.39).

The serum *Borrelia burgdorferi* s.l.–specific antibodies were positive by ELISA (Serion) and by immunoblot (anti-Borrelia Euroline RN-AT, Euroimmun) for IgG and IgM antibodies. On CSF *B. burgdorferi* s.l, antibodies were initially negative but became positive 3 weeks later. Intrathecal index was calculated and it was confirmed that there was blood–brain barrier disruption (=33.04 g/L; >2). Serum-specific bands on IgM (OspC Bg, OspC Bb and OspC Ba) and IgG (VlsE Ba, VlsE Bb, VlsE Bg and P41) immunoblot were observed, confirming the infection of *B. burgdorferi* s.l.

Serologic studies for *Mycoplasma* revealed a positive IgM with negative IgG, with no subsequent seroconversion and a negative CSF DNA amplification. The DNA amplification was initially positive for HHV-7, but negative on follow-up evaluation.

The patient was treated with intravenous ceftriaxone (2 g daily) for 3 weeks, with gradual clinical recovery within the first month. Six months after therapy, a follow-up spinal cord MRI showed improvement markedly.

DISCUSSION

In children, Lyme myelomeningoradiculitis is rare. Acute myelitis represents 4–5% of adult neuroborreliosis cases. The predominant clinical manifestations are upper or lower limbs weakness with arreflexia and bladder dysfunction, which did not occur in our patient.[2,3] However, 2 cases of paucisymptomatic extensive myelitis in children have already been reported.[3,4] Our patient also presented with a gradual, shingles-like pain because of painful meningopolyradiculitis. Spinal cord MRI is often nonspecific. However, it may demonstrate areas of edema, increased T2-weighted images and gadolinium enhancement of spinal cord and leptomeninges. Usually >3 metamers are involved mostly cervical, high thoracic or cervico-thoracic spine, as observed.[2,3]

Neuroborreliosis diagnosis is often difficult in low prevalence areas, such as Portugal where the estimated incidence is 0.04 per 100,000 inhabitants.[5,6] In Moldova, where our patient had stayed 3 months earlier, the incidence of Lyme borreliosis is significantly higher (0.73 per 100,000).[6] Intrathecal borrelial antibody production, isolation of *Borrelia* or DNA amplification from CSF samples is suggestive of CNS infection.[7] Yet, CSF DNA detection and culture have a low sensitivity yield and although blood seroconversion confirms recent borrelial infection, it does not support CNS involvement. At the time of the first lumbar puncture, our patient reported symptoms for >6 weeks, which do not explain the first negative CSF antibodies. However, the subsequent positive CSF/blood index in the second lumbar puncture confirmed the diagnosis. Possibly the symptoms duration, due to its vague nature, could have been emphasized by our patient. Although culture was negative, *Borrelia garinii* genospecies could probably be implicated. In fact, it is the most frequent European strain associated with neuroborreliosis, presenting more frequently with radicular pain and meningeal signs.[3]

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MYCOBACTERIUM BOVIS ABDOMINAL TUBERCULOSIS IN A YOUNG CHILD

Beatriz Pemartín, MD, Maria Portolés Morales, MD,† Maria Elena Carazo, MD,* Alfredo Marco Macián, MD,* and Ana Isabel Piqueras, MD**

Abstract: Abdominal tuberculosis can mimic many processes resulting in delayed diagnosis and unnecessary surgery. We describe a child of Moroccan parents with an abdominal mass caused by Mycobacterium bovis. The case stresses the need for increased awareness of *M. bovis* tuberculosis (TB) in general and extrapulmonary TB in the setting of foreign-born individuals who travel periodically to TB-endemic regions.

Key Words: Abdominal tuberculosis, abdominal mass, *Mycobacterium bovis*

From the *Department of Pediatric Surgery, and †Department of Pediatrics, Hospital La Fe, Valencia, Spain.

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Address for correspondence: Beatriz Pemartín, MD, Department of Pediatric Surgery, Hospital universitario y politécnico La Fe, c/Fernando Abril Martorell CP 46026, Valencia, Spain. E-mail: beapemartin@gmail.com.

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Abdominal tuberculosis (TB) can mimic many processes resulting in delayed diagnosis and unnecessary surgery. We describe a child of Moroccan parents with an abdominal mass caused by *M*ycobacterium *bovis*. The case stresses the need for increased awareness of *M. bovis* TB in general and extrapulmonary TB in